Cobalt Tumor of the Thyroid Gland

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COBALT is reported useful in therapy of certain kinds of anemia.[1, 2, 4, 9, 13, 17, 18] It may cause nausea, vomiting and loose stools, not severe enough to contraindicate its use. But goitrogenic effect, recently reported by Gross and Kriss with Spaet[6] and with Carnes[12] may be more serious. Pronounced hyperplasia of the thyroid gland was noted histologically. Clinically hypothyroidism was observed in some cases along with lowered metabolic rate and lessened iodine uptake. The goitrogenic effect has since been observed by others.[3, 10, 14] Even so, some manufacturers of hemotinics continue to recommend routine use of cobalt including during the latter part of gestation, without mentioning this goitrogenic effect.

Recently the authors removed a thyroid tumor (hyperplasia) which developed during iron and cobalt therapy in one of a pair of 8-month-old twins.

REPORT OF A CASE

In *Twin A*, a girl eight months old, a lump developed in the thyroid isthmus after two months of therapy for microcytic anemia with regular daily use of a cobalt-iron mixture (Roncovite®). The amount used daily was 1.2 ml., carrying about 20 mg. of cobalt. The hemoglobin value increased from 50 per cent up to 80 per cent during therapy but the mother had begun to notice the baby had difficulty with eating. In ten days a lump in the neck increased from about 2 cm. to 3 cm. in diameter and the patient was able to swallow only a few sips of liquid a day. Lugol's solution, 3 minims a day, did not make the mass recede.

In *Twin B*, a sister of Twin A, similar anemia had developed; the same therapy had been given and definite thyroid enlargement developed—diffuse but not great, and without nodule.

The twins were born six weeks prematurely. The mother had been seeking pregnancy for nine years. Conception occurred two months after diagnosis of hypothyroidism and the beginning of administration of desiccated thyroid, 60 mg. a day. Twin B, the first born, presented normally, weighed 4 pounds 6 ounces and was always the more robust. Twin A weighed 3 pounds 8 ounces and was a breech presentation. She had frequent respiratory infections, for which antibiotics had been given. The twins both have type 0, Rh (C De/Ce).

Upon examination, Twin A was observed to be undernourished (weight 15½ pounds), listless and sallow. The mass at the midline of the neck was rather firm. No other thyroid tissue could be felt. Offered water, the patient made rather exaggerated gulping motions, but swallowed only very small amounts. Breathing appeared unimpeded, although the mother had thought it noisy the night before.

Operation was done to remove the mass, which proved to be mostly thyroid isthmus. There was considerable tracheal compression. Sections taken from adjacent parts of the right and left lobes showed the same structure as the main mass—extreme hyperplasia throughout, and in some areas so great as to represent a papillary adenoma. No colloid was seen in any sections. The entire thyroid gland appeared about four times normal size, and it was extremely vascular. The stroma was dense in places, delicate in others (Figures 1 and 2).

Two days after operation, therapy with desiccated thyroid was started, 6.0 mg. a day, and administration of Lugol's solution was resumed. The patient did well. A month later, without consulting a physician, the mother began to give Twin A the cobalt-iron medication again, at the same time continuing the Lugol's solution and desiccated thyroid. For a while the baby continued to do well, but after a few weeks was taken to the family physician because of rapid thyroid enlargement, this time of the right lobe. She was again having trouble with eating. The tumor was spherical, firm, about 3 cm. in diameter. The patient had lost weight and become listless again.

Because of the similarity to the reported cases of thyroid hyperplasia during cobalt administration, the present cases were discussed with Dr. Ruth Gross,[5] who also thought cobalt might be the cause. All medication was stopped and five days later the tumor was definitely smaller. In two weeks it was almost gone, and the patient was eating well again. Observed for two months after discontinuance of cobalt, she seemed normal. A recent test with I^{131} showed 32 per cent uptake in the neck at 24 hours.

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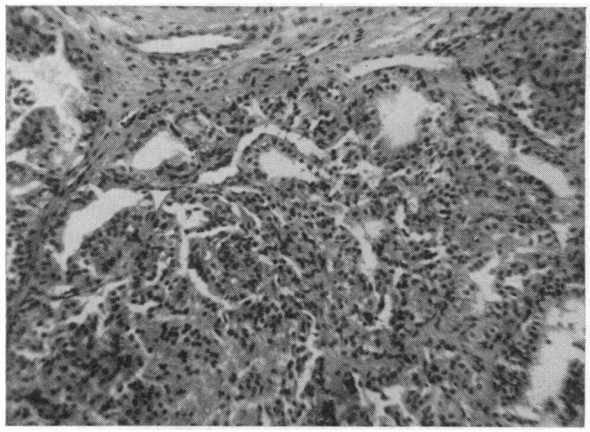

Figure 1.—Histological section (×100) of thyroid gland (Twin A) showing pronounced hyperplasia and absence of colloid.

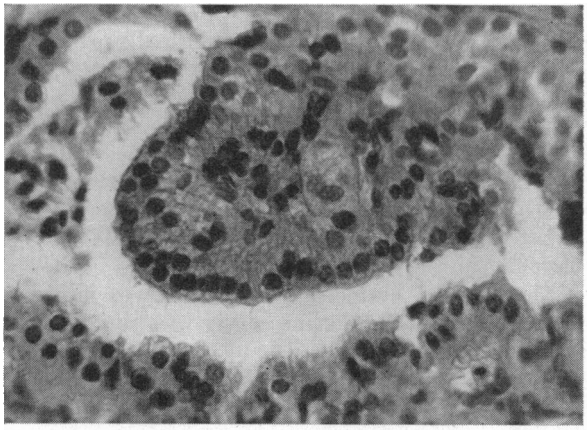

Figure 2.—High power photomicrograph (×400) of same specimen as shown in Figure 1.

Twin B had her medication stopped at the same time and there was a similar total regression of the (slight) thyroid enlargement, and a normal uptake of I^{131} (30 per cent at 24 hours) a month after discontinuance of therapy.

COMMENT

Thyroid tumor developing in association with cobalt administration has been reported infrequently, and only in children. Kriss, Carnes and Gross[12] reviewed autopsy material and noted one case in an adult. However, thyroid hyperplasia (from whatever cause) can cause death by suffocation. Klink[11] has reported ten such cases, five of them in infants that had received cobalt.

In the cases reported[6, 12] the tumors developed over a number of weeks (as in the case herein) and diminution in iodine uptake occurred at the same time. (This could not be tested in the patients herein reported upon, because of the use of Lugol's solution.) In the other reported cases, the tumors regressed after discontinuance of cobalt therapy, as happened in the patients in the present case.

Anemia was present in the patients here reported upon, and this was true of the other cases in which a goitrogenic effect was reported. Jaimet and Thode[8] reported upon 18 children who received cobalt without development of thyroid hyperplasia or decrease in I^{131} uptake. However—and perhaps this is significant—these children did not have anemia. One wonders if the susceptibility to the goitrogenic effect of cobalt is in some way related to certain types of anemia. Even though Holly[7] reported upon a series of 78 women receiving cobalt during gestation without enlargement of the thyroid gland and without abnormality of the baby, the authors feel that not enough is known as yet of the effect of cobalt on the fetal thyroid gland to warrant recommending its use in pregnant women.

CONCLUSION

Cobalt by mouth can cause dangerous hyperplasia of the thyroid gland, at least in infants and children. This can occur even during medication with thyroid and iodine. It is not certain what anemia has to do with the individual susceptibility to this effect. The thyroid enlargement regresses after cessation of cobalt medication. Thyroid function may then be found clinically normal, with normal uptake of iodine. The hazards should be carefully considered before cobalt is given to infants, children or pregnant women.

SUMMARY

Twin sisters eight months old had thyroid enlargement while receiving iron-cobalt medication (Roncovite). The enlargement was a single nodule in one, and operation was done to relieve obstruction to swallowing. A second tumor appeared in a few weeks on resumption of the use of Roncovite, although the patient was receiving Lugol's solution and desiccated thyroid at the time. This disappeared without operation after all medication was stopped. The diffuse thyroid enlargement in the other twin also disappeared after discontinuance of medication. Neither twin appeared clinically hypothyroid. Both had normal uptake of I^{131} in the neck when last observed.

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REFERENCES

1. Berk, L., Burchenal, J. H., and Castle, W. B.: Erythropoietic effect of cobalt in patient with or without anemia, New Eng. J. Med., 240:754, 1949.

2. Cartwright, G. E.: Dietary factors concerned in erythropoiesis, Blood, 2:256, 1947.

3. Gairdner, D., Marks, J., and Roscoe, J. D.: Goitrogenic hazard of cobalt, Lancet, 2:1285, 1954.

4. Gardner, F. H.: The use of cobaltous chloride in the anemia associated with chronic renal disease, J. Lab. Clin. Med., 41:56, 1953.

5. Gross, R. T.: Personal communication.

6. Gross, R. T., Kriss, J. P., and Spaet, T. H.: Hematopoietic and goitrogenic effects of cobaltous chloride in

patients with sickle cell anemia, Am. J. Dis. Child., 88:503, 1954.

7. Holly, R. G.: Studies on iron and cobalt metabolism, J.A.M.A., 158:1349, 1955.

8. Jaimet, C. H., and Thode, H. G.: Thyroid function studies on children receiving cobalt therapy, J.A.M.A., 158:1353, 1955.

9. Kato, K.: Iron cobalt treatment of physiologic and nutritional anemia in infants, J. Pediat., 11:385, 1937.

10. Keitel, H. G.: Cobalt and thyroid dysfunction, J.A.M.A., 158:1390, 1955.

11. Klink, G. H.: Thyroid hyperplasia in young children, J.A.M.A., 158:1347, 1955.

12. Kriss, J. P., Carnes, W. H., and Gross, R. T.: Hypothyroidism and thyroid hyperplasia in patients treated with cobalt, J.A.M.A., 157:117, 1955.

13. Robinson, J. C., James, G. W. III, and Kark, R. M.: Effect of oral therapy with cobaltous chloride on blood of patients suffering with chronic suppurative infection, New Eng. J. Med., 240:749, 1949.

14. McBryde, A. G.: Commenting on paper of Dr. Gross,[6] Am. J. Dis. Child., 88:503, 1954.

15. Scott, K. G., and Reilly, W. A.: Cobaltous chloride and iodine metabolism of normal and tumor-bearing rats, J.A.M.A., 158:1355, 1955.

16. Toxic effects of cobalt, Brit. M. J., 1:1331, 1955.

17. Wintrobe, M. M., Grinstein, M., Dubash, J., Humphreys, S., Ashenbrucker, H., and Worth, W.: The anemia of infection. VI. Influence of cobalt on the anemia associated with inflammation, Blood, 2:323, 1947.

18. Wolf, J., and Levy, I. J.: Treatment of sickle-cell anemia with cobalt chloride, Arch. Int. Med., 93:387, 1954.

Acute Urinary Retention in Pregnancy

Report of a Case

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INABILITY OF A PATIENT to evacuate the bladder adequately during pregnancy has been reported only in the situation of a retroverted uterus tightly wedged between the sacrum and pubis in such a manner that the uterine cervix forms a point of occluding pressure against the urethra and bladder neck. This complication of pregnancy has undoubtedly occurred many more times than the rather small number of reported instances in the medical literature would indicate. Nevertheless it is important to report a new instance of the condition because of the acute distress it causes, and for the light that can be cast on methods of treatment. Further, the fact that the condition might be confused with an abdominal condition requiring surgical intervention warrants the reporting of an additional case.

In the management of this problem, success has been reported with the use of two conservative procedures. Replacement of the uterus in the anterior position, with or without the subsequent use of a pessary, resulted in success without interruption of pregnancy in seven patients reported upon by several observers.[1,2,3]

In the present case, the acute emergency was abated without damage to the fetus, solely by vesical decompression and allowing the catheter to remain in place until the uterus spontaneously assumed the anteverted and elevated position as gestation progressed.

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REPORT OF A CASE

The patient, a 28-year-old white woman, was first observed in the first trimester of pregnancy. She complained of severe, dull, cramping pain in the lower abdomen of 12 hours' duration and said she had not passed urine for some 12 to 18 hours. She had had an appendectomy some ten years previously. The patient had two children who were delivered without untoward difficulty after normal pregnancy, and she had had no abortions or premature terminations of pregnancy.

The first day of the last menstrual flow was April 23, 1954. The patient was 5 feet 2 inches tall and weighed 132 pounds. The body temperature was 99.0° F. The radial artery pulse rate was 86 per minute and the blood pressure was 120/70 mm. of mercury.

There was a cystocele and rectocele of minimal degree present. The uterine cervix was blue, and on it was an area of erosion. The uterus was retroverted and enlarged to the size consistent with three months' gestation. The bladder was greatly distended and tender.

A No. 16 (French calibration) catheter with a Foley type balloon of 5 cc. capacity was inserted into the bladder. In the succeeding few minutes 1,600 cc. of urine was drained from the bladder. The catheter balloon was then expanded with water, and the catheter was left in the bladder.

Upon examination of the pelvic organs it was observed that the uterus was retroverted and incarcerated in the pelvis. No attempt was made to replace the uterus. Sulfasoxizole, 0.5 gm. three times daily, was prescribed. Two weeks later there was a small amount of bleeding, apparently of uterine origin, but it ceased spontaneously. At that time the uterus was of the size consistent with three and a half months' gestation. Twenty-three days after the onset of the acute urinary retention the patient reported that simultaneously the catheter was extruded spontaneously and "something moved" and the abdomen felt "different." Upon examination, the uterus was observed to be of a size consistent with four to four and a half months' gestation, and it was completely out of the cul de sac of Douglas and was anteverted.

There was no further difficulty with the pregnancy, and on February 3, 1955, the patient was delivered of a normal baby.